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Lecture

Melancholia and Depression During the 19th Century: A Conceptual History

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Melancholia before the 19th century

The meaning of 'melancholia' in classical antiquity is opaque and has little in common with 20th-century psychiatric usage (Drabkin, 1955; Heiberg, 1927). At that time, melancholia and mania were not polar opposites (i.e. one was not defined as having opposite features to the other). Melancholia was defined in terms of overt behavioural features such as decreased motility, and morosity (Roccatagliata, 1973; Simon, 1978). Hence, in medical usage, 'melancholia' referred to a subtype of mania and named, in general, states of reduced behavioural output. These included disorders that might "exhibit depressed, agitated, hallucinatory, paranoid and even demented states . . . the ancient diagnosis of melancholy has no correct analogue in modern psychiatric practice . . ." (Siegel, 1973, p. 274).

Historical semantics shows that symptoms reflecting pathological affect (e.g. sadness) were not part of the concept, although, occasionally, reference to such might have been made (Berrios, 1985*a*). Writing on this very issue, more than 150 years ago, Prichard (1835) reached the same conclusion ". . . Anciently this word, in ordinary language at least, conveyed no idea of gloom or dejection. Melancholic meant simply to be mad" (p. 27).

The meaning of melancholia, in fact, can only be understood in terms of the humoural theory (Tracy, 1969). This generated predictions as to the behaviour of the subject and his or her response to treatment (Flashar, 1966; Walser, 1968; Starobinski, 1962). As often, Jones (1972) got it right: "the word is closely connected both with the doctrine of the humours and with the prevalence of malaria . . . in popular speech melancholia and its cognates sometimes approximate in meaning to 'nervous breakdown'. Probably the name was given to any condition resembling prostration, physical and mental, produced by malaria, one form of which (the quartan) was supposed to be caused by 'black bile' ('melaina kole')" (p. lviii).

There seems to have been little change in the meaning of melancholia during the Middle Ages (Leibbrand & Wettley, 1961; Jacquart, 1983) in spite of the fact that, during this period, a harder and more practical view of madness than had hitherto been considered was taken (Kroll & Bachrach, 1984). It has been claimed, however, that by the time of Galen, the notion of melancholia had moved closer to current definitions (Jackson, 1969, p. 375). The development of concepts such as 'nostalgia' (Rosen, 1975; Bégin, 1834; Rauchs, 1985) and 'melancholy' helped to form a family of terms in which affective symptoms were emphasised (Jackson, 1981, 1983, 1986).

Occasional references to states combining mania and melancholia were made before the 19th century (Briand & Azemar, 1923), but in terms of the nosographic beliefs entertained in these earlier periods, it makes little sense to talk about real 'anticipations' of manic–depressive psychosis (Huber, 1985) (for a history of mania, *see* Berrios, 1988*a*).

The theoretical background

The transformation of the old category of melancholia into its current counterparts did not occur in a vacuum. Changes in the concept of disease, in the psychological definition of behaviour, and in taxonomic principles were needed. All three changes occurred during the early 19th century (Berrios, 1988*b*).

Concept of disease

The so-called clinico-anatomical view, developed during the early 19th century, was that overt signs of illness could be correlated (without residuum) with anatomical lesions (López Piñero, 1983; Ackerknecht, 1967; Laín Entralgo, 1978). Anatomical lesion, in turn, was defined in terms of tangible units of analysis. These were to change during the 19th century from organ to tissue and finally to cell. Frequent failure to identify such lesions led, during the second half of the 19th century, to the

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redefinition of 'lesion' in physiological terms. This made possible, *inter alia*, the incorporation of the neuroses into psychiatry (López Piñero, 1983).

The implementation of the clinico-anatomical view required the creation of a descriptive language (Berrios, 1984). Although based on observation, the latter included some conceptual assumptions. The most important referred to the legitimacy of sectioning mad behaviour into well-defined categories. The monolithic categories of the 1800s were thus gradually transformed; some disappeared altogether, others were reclaimed and refurbished with different meaning (e.g. melancholia), others were left intact (e.g. delirium) (Berrios, 1981*a*).

Psychological definition of behaviour

Symptom descriptions were made possible by the availability of new psychological theories (Berrios, 1988*b*), and the alienist could then map and define behaviour, and reclaim subjective experience as another source of symptoms (Moreau de Tours, 1859).

Up to the end of the 18th century associationism was the predominant psychological theory in Europe (Warren, 1921; Hoeldtke, 1967). It had provided the new science, since the times of Hobbes and Locke, with its epistemological model. But at the end of the 18th century, faculty psychology experienced a rebirth, particularly in the work of Kant, Reid, and Stewart (Berrios, 1988*b*). The suggestion that the mind was a collection of functionally autonomous modules provided 19th century alienists with a useful classificatory framework. For example, Pinel (1809) and Prichard (1835) abandoned the intellectualistic view of madness generated by Lockean associationism and turned to faculty psychology.

The governing taxonomic principle during the 19th century was the possibility that the three clusters of mental functions (intellectual, emotional, and volitional) could become diseased separately. Thus the 'intellectual insanities' became the embryos of schizophrenia and paranoia, the emotional insanities of depression and mania, and the volitional insanities, of the psychopathic disorders (Berrios, 1988*b*).

Taxonomic principles

The philosophy of medical taxonomy also changed during the early 19th century. The botanical schematas of Linne, Sauvage, Cullen, and others (Bowman, 1975; Larson, 1971) were replaced by empirical principles (Desruelles *et al*, 1934; Baillarger, 1853; Foville, 1872; Vié, 1940). These originated from symptom comparison and frequential distribution, and even aetiological speculation. A new criterion, the natural history of the disease (i.e. the 'time' dimension), was only fully incorporated in psychiatry towards the end of the century (Del Pistoia, 1971; Lanteri Laura, 1972).

Taxonomic efforts during the 19th century were bedevilled by the limited success of the 'organic view' (failure due, in all likelihood, to limited technological resources) and by excessive theoretical speculation. For example, the abuse of the genetic explanation led to the so-called 'degeneration theory', which turned out to be a 'blind alley' (Morel, 1857; Walter, 1956; Wettley, 1959; Friedlander, 1973; Danion *et al*, 1985; Dowbiggin, 1985).

One of the interesting offshoots of the taxonomic controversy was the development of the unitary, and the multiple, views of insanity. Inspired by a theoretical principle (the indivisibility of the mind), and driven by exasperation with contemporary classifications, a group of alienists put forward, during the middle of the 19th century, the view that there was only one form of insanity and that its multiple clinical presentations resulted from idiosyncratic or pathoplastic factors (Llopis, 1954; Menninger *et al*, 1958; Rennert, 1968; Vliegen, 1980; Beer & Berrios, in press). It was suggested, for example, that mania, melancholia, delusional insanity, and vesanic dementia were but successive stages of the same disease. This is a rather important point to remember when the question of the circular insanity (bipolar disorder) is analysed, because the proposal that mania and melancholia must be related was based not only on clinical observation (Sedler & Dessein, 1983) but also on the at-the-time popular view that all forms of insanity related to one another.

Melancholia during the 19th century

"Melancholia", wrote John Haslam in 1809, "the other form in which this disease (madness) is supposed to exist, is made by Dr Ferriar to consist in 'intensity of idea'. By intensity of idea I presume is meant, that the mind is more strongly fixed on, or more frequently recurs to, a certain set of ideas, than when it is in a healthy state . . ." (pp. 32–33). This perception was correct. Up to the dawn of the 19th century, forms of behaviour that (from our perspective) had little in common were included under the general class, melancholia (Madden, 1966). There is no better example of this than the contents of Burton's *Anatomy of Melancholy* (1883). Sadness can occasionally be found among these symptoms but was not used for definition in any real sense (Lewis, 1934).

That a semantic break in the meaning of melancholia must have taken place during the early 19th century is not a recent suggestion. Esquirol (1820) sensed it: "the word melancholia, consecrated in popular language to describe the habitual state of sadness affecting some individuals should be left to poets and moralists whose loose expression is not subject to the strictures of medical terminology" (p. 148). Prichard (1835) had a similar view, and Rush (1812), after criticising Cullen's usage, also advised against the use of the word 'melancholia' and coined a new term 'tristimania'.

The 'lypemania' concept

As mentioned above, faculty psychology provided a taxonomic matrix for 19th-century psychiatry. Disease had been classified until then according to the *more botanico* tradition (López Piñero, 1983) in terms of privileged 'features' such as aetiology (e.g. Battie, 1758) or behavioural forms (e.g. Crichton, 1798; Arnold, 1782). The classification of melancholia included symptoms other than emotional disturbance; in fact, it was defined as a disease of 'partial delusions'. This 'intellectualistic' origin disqualified melancholia from being the right name for the new 'emotional insanities'.

Thus Esquirol (1820) was forced to coin the word 'lypemania' to refer to: "a disease of the brain characterised by delusions which are chronic and fixed on specific topics, absence of fever and *sadness which is often debilitating and overwhelming*. It must not be confused with mania which exhibits generalised delusions and excited emotions and intellect nor with monomania that exhibits specific delusions and expansive and gay emotions, nor with dementia characterised by incoherence and confusion of ideas resulting from weakening . . ." (pp. 151–152, my italics).

Esquirol (1820) reported statistical findings on his new disease. Admission rates for lypemania were found to be increased between May and August (p. 159), the most affected age group was 25–45 (p. 161), in 110 of 482 cases 'heredity' seem to play a role, and its common causes included domestic crisis, grief, and disturbed relationships (p. 166). About a third of his cohort died, often of tuberculosis.

Unfortunately, the term lypemania proved to be, as Delasiauve (1856) put it, too "élastique . . . apart from being less imprecise was no different in terms of contents from the old word melancholia" (p. 382). Delasiauve was here criticising the fact that 'partial' delusions (a vestige of the old intellectualistic notion) had remained a defining criterion of lypemania. He suggested that the meaning of lypemania was narrowed further to refer to: "an exaggeration and persistence of feelings of depression" (p. 384). Delasiauve went on to attack Baillarger for considering 'stupidité' (stupor) and lypemania (rightly as it turned out to be!) as varieties of the same disease (p. 441) (Berrios, 1981*b*).

The highest point in the history of the lypemania concept was reached in the work of Billod (1856), who attempted a classification and a further refinement of its psychopathology. Billod accepted that lypemania had to be defined on the basis of sad delusions and affect, and suggested a fourfold classification. There was lypemania with: sad delusions and sadness; sad delusions and no sadness; sad delusions and mixed or alternating affective disorder (this included the bipolar states); and no sad delusions and sadness. This contrived and symmetric classification allowed the recognition of about 16 clinical subtypes. Some of these have since disappeared (e.g. ironic or religious lypemania) but others (e.g. hypochondriacal, stuporous, or irritable lypemania) are still recognisable, although they now have different names.

Apart from in France, the term lypemania was only used in Spain. It never 'caught on' in Germany, Austria, Switzerland, or the UK, where the word 'melancholia' was maintained. Prichard (1835) paid no attention to the term; nor did Griesinger (1867) who, although quoting Esquirol often enough, did not take notice of his neologism. Feuchtersleben (1847) quoted the term once but did not acknowledge its origins. Bucknill & Tuke (1858) did, but continued using melancholia on the excuse that Esquirol himself had stated that the two terms could be used interchangeably (p. 147). 'Mental depression' gradually replaced it towards the end of the century, and 'melancholia' was more and more used to name a subtype of psychotic depression occurring in the elderly (Chaslin, 1912). The word lypemania should be considered as a good historical example of how 'bridge' categories rarely survive. After effecting the transition of the concept of melancholia from the intellectualistic to the emotional, it quietly died away.

The term 'depression'

Delasiauve's quotation contains one of the earliest technical uses of the word 'depression'. By 1860, however, the word is already found in medical dictionaries: "applied to the lowness of spirits of persons suffering under disease" (Mayne, 1860, p. 264). It is important to ask why this term was needed. It seems to have suggested both a physiological and metaphorical 'lowering' of emotional function and hence had the semantic capacity to

name either a 'symptom' or a condition. In this way, the earlier theoretical views of Griesinger (1867) and the folkloric descriptions of melancholia (as sadness) could be reconciled. This is well illustrated in the first (1885) – of many – editions of Régis's Manual, where depression is defined as: "the state opposed to excitation. It consists in a reduction in general activity ranging from minor failures in concentration to total paralysis . . ." (p. 77).

General physicians seem also to have preferred depression to melancholia or lypemania, perhaps because the term evoked a 'physiological' explanation. Sir William Gull (of anorexia nervosa fame) used it as early as 1868 in his classical article on "hypochocondriasis"; "its principal feature is mental depression, occurring without apparently adequate cause . . ." (Gull, 1894, p. 287). By the end of the century, 'depression' had become a synonym of melancholia: "a condition characterised by a sinking of the spirits, lack of courage or initiative, and a tendency to gloomy thoughts. The symptom occurs in weakened conditions of the nervous system, such as neurasthenia and is specially characteristic of melancholia" (Baldwin, 1901, p. 270). Savage (1898) in his very popular *Insanity and the Allied Neuroses* (studied by many generations of clinical students in the UK) defined melancholia as "state of mental depression, in which the misery is unreasonable . . ." (p. 151).

In all three quotations, the term 'depression' refers to a symptom. Kraepelin (1921), however, used 'depressive states' as a generic category under which he included melancholia simplex, stupor, melancholia gravis, fantastic melancholia, and delirious melancholia. In the UK, this group of disorders continued to be classed as 'melancholia'. Thus, in the famous *Nomenclature of Diseases* drawn up by a Joint Committee appointed by the Royal College of Physicians of London (1906), melancholia was classified as a "disease of the nervous system" (code 146), exhibiting acute, recurrent, or chronic states. The Committee advised that "the variety when known should be returned according to the following categories: agitated, stuporous, hypochondriacal, puerperal, climacteric, senile, and from acute or chronic disease, or from injury' (p. 37).

Endogenous depression and melancholia

The qualifier 'endogenous', introduced at the turn of the century, has not been particularly enlightening (Masi, 1981). Conceptually based on the old 'degeneration theory', it has carried a semantic contraband that cannot be accommodated in the 20th century. Kraepelin proposed a division of mental diseases into exogenous and endogenous and acknowledged his debt to Möbius (1893). The word 'endogenous' (originally coined in the context of botanic classification by De Candolle in 1813; Heron, 1965) was used in *fin de siècle* psychiatry as a 'technical' term to refer to psychopathological states believed to result from the 'degeneration' of the human seed (e.g. hysteria and manic–depressive illness) (Lewis, 1971). The rest of the psychiatric disorders were 'exogenous'. The dividing line was not drawn between environment and the body (as it is often assumed) (Berrios, 1987), but between illness stemming from the Anlage (abstract concept that included both genetics and psychogenicity) and the rest of possible causes (Schiller, 1982).

The combined states

Clinical historians cannot help 'mining' for references that prove that in the remote past someone 'discovered' manic–depressive illness, and Aretaeus the Capadoccian has often been granted the dubious honour (Kotsopoulos, 1986; Jellife, 1931). These efforts are of little use as the concept of disease that made the concept of bipolar disorder possible only developed during the 19th century.

The conceptual conditions required for the notion of alternating insanity to emerge were: 1. the consolidation of the clinico-anatomical view of disease; 2. a longitudinal as opposed to a cross-sectional definition of disease (the introduction of this principle we owe to Kahlbaum and not Kraepelin) (Berrios & Hauser, in press); 3. stability in the semiology of affectivity, which included codification of features such as regularity, intensity, congruity, rhythm, etc. (Berrios, 1985*a*); 4. availability of a concept of personality; and 5. strategies for differential diagnoses (e.g. *per genus et differentiae*).

All five conditions were met after 1850. Almost immediately after the circular insanities were described, Falret (1854) and Baillarger (1854) embarked on a protracted dispute over priority, but with the benefit of hindsight, it seems clear that both described uncommon (and rigid) evolutionary patterns for the disease. Falret insisted that mania and melancholia alternated at regular intervals; and Baillarger that between the two there was an (intercalated) third period of lucidity.

European psychiatry soon took to the condition. In 1880 the French Academy of Medicine opened a competition on it (won by A. Ritti), and the same year the English Committee in charge of the organisation of the London Psychiatric Meeting of 1881 included a session to discuss the clinical relevance of the new disease.

To become more acceptable and clinically useful, the illness needed to escape from the rigid alternations suggested both by Falret and Baillarger. This was achieved during the 1880s when the concept of alternating 'personality' (an attenuated form of the disease) became fully recognised, as did its genetic basis. Various patterns and combinations were described with regards to the intensity, duration, and alternation of the three basic states (mania, melancholia, and lucidity).

There was, however, some debate as to whether the combination was coincidental and after all, no real alternating disease existed (Morel and Dagonet), or whether, if it did exist, the isolated presentation of mania or depression were in fact *formes frustres* of the total disease (Marce, Foville, Ritti Luys, Billod, Meyer, Kirn, Krafft Ebing, and Karrer). Indeed, efforts were made to identify clinical features that could allow diagnosis on the cross-sectional examination, thus obviating the need for longitudinal observation.

Differential diagnosis was made with pure mania, lypemania (depression) and the 'exalted state' of the general paralysis of the insane. There is no space in this short paper to chronicle this debate in detail. An important clinical issue was, for example, the observation that while pure mania was more often characterised by thought disorder and delusions, mania (as part of a combined state) showed more often hyperactivity and elation (delusions of affect, movement, and action as the French called it) (Foville, 1882). This interesting clinical hypothesis has not been fully tested.

The aftermath

Kraepelin (1921) solved the differential-diagnosis problem by claiming that there were no real differences between all these states: "manic depressive insanity as it is to be described in this section, includes on the one hand the whole domain of so called periodic and circular insanity, on the other hand simple mania, the greater part of the morbid states termed melancholia and also a not inconsiderable number of cases of amentia" (p. 2). Whether this unitary view is right still remains to be seen, but those who, to this day, defend the Kraepelinian integration must be reminded that "amentia" (a concept described by Meynert, 1890) should not be quietly forgotten simply because it might prove a clinical embarrassment. Historical analysis shows that Kraepelin might, after all, have been right in his inclusion, as amentia also referred to states of depressive and manic pseudodementia (Berrios, 1985*b*; Bulbena & Berrios, 1986). Kraepelin's integration was challenged by major figures of European psychiatry who wanted to keep the melancholias (unipolar depressions) in a separate group. A typical representative of this group was Chaslin (1912).

Conclusions

This short paper has dealt with the conceptual history and ideological background that made possible the transformation of the pre-19th century notion of melancholia into the concepts of depression and bipolar disorder, from the medical and psychological changes operating at the beginning of the 19th century to Kraepelin's integrative work. The old notion of melancholia was refurbished with meaning and its transition to depressive illness was facilitated by Esquirol's concept of lypemania, which, for the first time, emphasised the primary affective nature of the disorder. Finally, once the right conceptual conditions had obtained, melancholia and mania were combined into the concept of alternating, periodic, circular, or double-form insanity, the earlier rigid-pattern descriptions of the disease were rendered flexible, this process culminating with Kraepelin's final synopsis.

The 19th century built six theoretical principles into our current concept of manic–depressive illness: it was a 'primary' disorder of affect, and not of intellect or cognition (Bolton, 1908); it had stable psychopathology (Foville, 1882); it had brain representation (Ritti, 1876); it was periodic in nature (Falret, 1854; Baillarger, 1854); it was genetic in origin (Foville, 1882); and it tended to appear in individuals with recognisable personality predisposition (Ritti, 1876). The real causes of the episodes remained endogenous in nature (Chaslin, 1912).

All these features were extracted from clinical observation, descriptive statistics, and logical reasoning, and were based on 19th century assumptions as to how the normal and pathological mind worked. That we still share these assumptions is best illustrated by our still being trapped in what Kraepelin called the "circle" of the manic–depressive insanity.

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